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NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	AUG 10	Time limit for inactive STN sessions doubles to 40 minutes
NEWS	3	AUG 18	COMPENDEX indexing changed for the Corporate Source (CS) field
NEWS	4	AUG 24	ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
NEWS	5	AUG 24	CA/Caplus enhanced with legal status information for U.S. patents
NEWS	6	SEP 09	50 Millionth Unique Chemical Substance Recorded in CAS REGISTRY
NEWS	7	SEP 11	WPIDS, WPINDEX, and WPIX now include Japanese FTERM thesaurus
NEWS	8	OCT 21	Derwent World Patents Index Coverage of Indian and Taiwanese Content Expanded
NEWS	9	OCT 21	Derwent World Patents Index enhanced with human translated claims for Chinese Applications and Utility Models
NEWS	10	NOV 23	Addition of SCAN format to selected STN databases
NEWS	11	NOV 23	Annual Reload of IFI Databases
NEWS	12	DEC 01	FRFULL Content and Search Enhancements
NEWS	13	DEC 01	DGENE, USGENE, and PCTGEN: new percent identity feature for sorting BLAST answer sets
NEWS	14	DEC 02	Derwent World Patent Index: Japanese FI-TERM thesaurus added
NEWS	15	DEC 02	PCTGEN enhanced with patent family and legal status display data from INPADOCDB
NEWS	16	DEC 02	USGENE: Enhanced coverage of bibliographic and sequence information
NEWS	17	DEC 21	New Indicator Identifies Multiple Basic Patent Records Containing Equivalent Chemical Indexing in CA/Caplus
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=> s calcitonin gene related peptide or CGRP
L1 31928 CALCITONIN GENE RELATED PEPTIDE OR CGRP

=> s pituitary adenylate cyclase activating peptide or PACAP
L2 9041 PITUITARY ADENYLATE CYCLASE ACTIVATING PEPTIDE OR PACAP

=> s 11 or 12
L3 40576 L1 OR L2

=> s 13 and interstitial cystitis
L4 43 L3 AND INTERSTITIAL CYSTITIS

=> dup rem 14
PROCESSING COMPLETED FOR L4
L5 27 DUP REM L4 (16 DUPLICATES REMOVED)

=> d bib abs 1-

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L5 ANSWER 1 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

DUPLICATE 1

AN 2009:24949 BIOSIS

DN PREV200900024949

TI Functional and Immunohistochemical Characterization of CB1 and CB2

Receptors in Rat Bladder.

AU Hayn, Matthew H. [Reprint Author]; Ballesteros, Inmaculada; de Miguel,

Fernando; Coyle, Christian H.; Tyagi, Shachi; Yoshimura, Naoki; Chancellor, Michael B.; Tyagi, Pradeep

CS Univ Pittsburgh, Med Ctr, Dept Urol, Kaufmann Bldg, Suite 700, 3451 5th Ave,

Pittsburgh, PA 15213 USA

haynm2@upmc.edu

SO Urology, (NOV 2008) Vol. 72, No. 5, pp. 1174-1178.

ISSN: 0090-4295.

DT Article

LA English

ED Entered STN: 17 Dec 2008

Last Updated on STN: 17 Dec 2008

AB OBJECTIVES To determined the localization of CB1 and CB2 receptors in rat

bladder and investigate the effect of a mixed CB1/CB2 receptor agonist,

ajulemic acid (AJA), on chemically evoked release of the sensory neuropeptide calcitonin gene-related peptide (CGRP).METHODS

Whole rat bladders were incubated in a series of tissue baths containing

physiologic salt solution to measure baseline CGRP release by enzyme immunoassay. Capsaicin (30 nM) and adenosine

triphosphate (10 mu

M) were used to provoke CGRP release in the presence or absence of AJA. Specificity of AJA for CB1 and CB2 receptors was

determined using

antagonists. Localization was determined by immunofluorescence for CB1

and CB2 receptors in fixed bladders.RESULTS Immunofluorescence, showed the

localization of CB1 and CB2 receptors in the bladder. Mean baseline

CGRP release was 605 +/- 62 pg/g of bladder weight, and AJA had no

effect on CGRP release. The addition of adenosine triphosphate/capsaicin significantly increased the CGRP release over baseline, by 44% (P < .05), and AJA application

significantly

decreased CGRP release, by 29% compared with controls ($P < .05$).
The CB1 and CB2 antagonists AM 251 and AM 630, respectively,
reversed the blunting effect of AJA on evoked CGRP release, resulting in an
increase of 40% and 38% over baseline, respectively. CONCLUSIONS
CB1 and CB2 receptors are localized in the urothelium of rat bladder, and
application of AJA inhibits the evoked release of CGRP by acting
on CB1 and CB2 receptors. These findings identify a potential
new pathway for study in the evaluation and treatment of painful bladder
syndrome/
interstitial cystitis. UROLOGY 72: 1174-1178, 2008. (C)
2008 Elsevier Inc.

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AN 2008366272 EMBASE
TI Reply.
AU Liu, Hsin-Tzu, Dr. (correspondence); Kuo, Hann-Chorng
CS Department of Urology, Buddhist Tzu Chi General Hospital, Tzu Chi
University, Hualien, Taiwan, Province of China.
SO Urology, (August 2008) Vol. 72, No. 2, pp. 464.
Refs: 5
ISSN: 0090-4295; E-ISSN: 1527-9995 CODEN: URGYAZ
PB Elsevier Inc., 360 Park Avenue South, New York, NY 10010, United
States.
PUI S 0090-4295(08)00228-8
CY United States
DT Journal; Letter
FS 028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LA English
ED Entered STN: 4 Sep 2008
Last Updated on STN: 4 Sep 2008

L5 ANSWER 3 OF 27 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All
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reserved on STN
AN 2008366271 EMBASE
TI Intravesical Botulinum Toxin A Injections Plus Hydrodistension
Can Reduce
Nerve Growth Factor Production and Control Bladder Pain in
Interstitial Cystitis: A Molecular Mechanism.
AU Namazi, Hamid, Dr. (correspondence)
CS Department of Orthopaedic Surgery, Shiraz University of Medical
Sciences,
Chamran Hospital, Shiraz, Iran, Islamic Republic of.
SO Urology, (August 2008) Vol. 72, No. 2, pp. 463-464.
Refs: 6

ISSN: 0090-4295; E-ISSN: 1527-9995 CODEN: URGYAZ
 PB Elsevier Inc., 360 Park Avenue South, New York, NY 10010, United States.
 PUI S 0090-4295(08)00227-6
 CY United States
 DT Journal; Letter
 FS 028 Urology and Nephrology
 030 Clinical and Experimental Pharmacology
 037 Drug Literature Index
 LA English
 ED Entered STN: 4 Sep 2008
 Last Updated on STN: 4 Sep 2008

L5 ANSWER 4 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
 DUPLICATE 2
 AN 2008:134998 BIOSIS
 DN PREV200800124454
 TI Botulinum toxin type A inhibits sensory neuropeptide release in rat bladder models of acute injury and chronic inflammation.
 AU Lucioni, Alvaro [Reprint Author]; Bales, Gregory T.; Lotan, Tamara L.; McGehee, Daniel S.; Cook, Sean P.; Rapp, David E.
 CS Univ Chicago, Pritzker Sch Med, Dept Surg, Urol Sect, 5841S, Maryland Ave, MC 6038, Chicago, IL 60637 USA
 alvarolucioni@hotmail.com
 SO BJU International, (FEB 2008) Vol. 101, No. 3, pp. 366-370. ISSN: 1464-4096.
 DT Article
 LA English
 ED Entered STN: 20 Feb 2008
 Last Updated on STN: 20 Feb 2008
 AB To determine the effect of botulinum toxin type A (BTX-A) on the release of the neuropeptides substance P (SP) and calcitonin gene-related peptide (CGRP) from isolated bladder preparations after acute injury with HCl and the induction of cyclophosphamide (CYP)-induced cystitis, as neurogenic inflammation has been increasingly identified in urological disorders such as interstitial cystitis. Adult rats had either an intraperitoneal injection with CYP or saline over a 10-day period to induce chronic bladder inflammation, after which the bladder was harvested, or normal bladder explants were injured acutely with incubation (20 s) in HCl (0.4 M). To measure the effect of BTX-A on the release of neurotransmitters, harvested bladders were incubated in an organ bath

containing BTX-A (10 U) or vehicle. Bladders were transferred to a subsequent bath (physiological saline) and incubated for 15 min, and the bathing medium analysed to measure neurotransmitter release, as determined by radioimmunoassay. Bladder specimens from sham treatment, controls and experimental rats were compared histologically. Acute injury with HCl caused a significantly greater release of both CGRP and SP release (1235 and 1655 pg/g, respectively) than in controls (183 and 449 pg/g, respectively; $P < 0.001$). This increase in neurotransmitter release was partly inhibited by exposure to BTX-A (870 and 1033 pg/g ($P < 0.05$ and < 0.01). CYP-induced chronic inflammation caused significantly greater release of SP than in the controls (1060 and 605 pg/g, respectively; $P < 0.005$). Exposure to BTX-A partly inhibited the release of SP after CYP-induced cystitis (709 pg/g, $P < 0.05$). The application of BTX-A inhibits the release of sensory neurotransmitters from isolated bladder preparations in rat bladder models of both acute injury and chronic inflammation, suggesting a potential clinical benefit of BTX-A in the treatment of neurogenic inflammation.

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AN 2008555557 EMBASE

TI Inside information: The unique features of visceral sensation.

AU Robinson, David R.; Gebhart, G.F.

CS Department of Anesthesiology, Pittsburgh Center for Pain Research,

University of Pittsburgh, Pittsburgh, PA 15213, United States.

AU Robinson, D. R., Dr. (correspondence)

CS Department of Anesthesiology, Pittsburgh Center for Pain Research,

University of Pittsburgh, Pittsburgh, PA 15213, United States.

SO Molecular Interventions, (1 Oct 2008) Vol. 8, No. 5, pp. 242-253.

Refs: 76

ISSN: 1534-0384; E-ISSN: 1543-2548 CODEN: MIONAR

PB American Society for Pharmacology and Experimental Therapy, 9650 Rockville

Pike, Bethesda, MD 20814, United States.
CY United States
DT Journal; General Review; (Review)
FS 002 Physiology
008 Neurology and Neurosurgery
048 Gastroenterology
LA English
SL English
ED Entered STN: 19 Dec 2008
Last Updated on STN: 19 Dec 2008
AB Most of what is written and believed about pain and nociceptors originates from studies of the "somatic" (non-visceral) sensory system. As a result, the unique features of visceral pain are often overlooked. In the clinic, the management of visceral pain is typically poor, and drugs that are used with some efficacy to treat somatic pain often present unwanted effects on the viscera. For these reasons, a better understanding of visceral sensory neurons - particularly visceral nociceptors - is required. This review provides evidence of functional, morphological, and biochemical differences between visceral and non-visceral afferents, with a focus on potential nociceptive roles, and also considers some of the potential mechanisms of visceral mechanosensation.

L5 ANSWER 6 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2008:74342 BIOSIS
DN PREV200800073712
TI Repeated botulinum toxin injections: A new answer for further questions.
AU Lazzeri, Massimo [Reprint Author]
CS Dept Urol, Santa Chiara Firenze Giomi Grp, Pzza INdipendenza 11, I-50129 Florence, Italy
lazzeri.m@tiscali.it
SO European Urology, (DEC 2007) Vol. 52, No. 6, pp. 1571-1573.
CODEN: EUURAV. ISSN: 0302-2838.
DT Article
Editorial
LA English
ED Entered STN: 16 Jan 2008
Last Updated on STN: 16 Jan 2008

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AN 2007494550 EMBASE

TI Resiniferatoxin and botulinum toxin type A for treatment of lower urinary tract symptoms.

AU Cruz, Francisco, Dr. (correspondence); Dinis, Paulo

CS Department of Urology, Hospital de S. Joao, Faculty of Medicine/IBMC of Porto, Porto, Portugal. cruzfjmr@med.up.pt

AU Cruz, Francisco, Dr. (correspondence)

CS Department of Urology, Hospital de S. Joao, P-4200 Porto, Portugal.

cruzfjmr@med.up.pt

SO Neurourology and Urodynamics, (2007) Vol. 26, No. 6 SUPPL., pp. 920-927.

Refs: 57

ISSN: 0733-2467; E-ISSN: 1520-6777 CODEN: NEUREM

CY United States

DT Journal; Conference Article; (Conference paper)

FS 028 Urology and Nephrology

037 Drug Literature Index

006 Internal Medicine

008 Neurology and Neurosurgery

LA English

SL English

ED Entered STN: 23 Oct 2007

Last Updated on STN: 23 Oct 2007

AB Resiniferatoxin (RTX) and botulinum toxin subtype A (BTX-A) are increasingly viewed as potential treatments for lower urinary tract symptoms (LUTS) refractory to conventional therapy. RTX, a capsaicin analogue devoid of severe pungent properties, acts by desensitizing the transient receptor potential vanilloid type 1 (TRPV1) receptor and inactivating C-fibers. BTX-A cleaves soluble N-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) proteins in afferent and efferent nerve endings, therefore impeding the fusion of synaptic vesicles with the neuronal membrane necessary for the release of neurotransmitters.

In patients with neurogenic and idiopathic detrusor overactivity, RTX and BTX-A have been shown to increase the volume to first detrusor contraction, increase bladder capacity, and improve urinary incontinence and quality of life. Recent data also suggest a role for these neurotoxins in treating urgency, the primary symptom in overactive bladder

(OAB) syndrome. Furthermore, experimental data strongly support the use of both neurotoxins in the treatment of pain and frequency in patients with interstitial cystitis/painful bladder syndrome (IC/PBS), although the results from available clinical trials for this use are still inconclusive. In spite of promising results overall, it should be made clear that the administration of these neurotoxins is still considered an experimental procedure and that more clinical studies are necessary before a license for their use will be issued by health authorities. .COPYRG. 2007 Wiley-Liss, Inc.

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AN 2007245559 EMBASE

TI New insights into the pathogenesis of fibromyalgia syndrome: Important

role of peripheral and central pain mechanisms.

AU Staud, Roland (correspondence)

CS Division of Rheumatology and Clinical Immunology, McKnight Brain Institute, University of Florida, Gainesville, FL 32610-0221, United States. staudr@ufl.edu

AU Staud, Roland (correspondence)

CS Department of Medicine, University of Florida, College of Medicine,

Gainesville, FL 32610-0221, United States. staudr@ufl.edu

SO Current Rheumatology Reviews, (May 2007) Vol. 3, No. 2, pp. 113-121.

Refs: 143

ISSN: 1573-3971

CY Netherlands

DT Journal; General Review; (Review)

FS 026 Immunology, Serology and Transplantation

003 Endocrinology

031 Arthritis and Rheumatism

005 General Pathology and Pathological Anatomy

008 Neurology and Neurosurgery

LA English

SL English

ED Entered STN: 26 Jun 2007

Last Updated on STN: 26 Jun 2007

AB Clinical symptoms of chronic muscle conditions like fibromyalgia (FM),

include pain, stiffness, subjective weakness, and muscle fatigue. Pain in

FM is usually described as fluctuating and always associated with local or

generalized tenderness (hyperalgesia and/or allodynia). This tenderness related to FM pain depends on increased peripheral and/or central nervous system responsiveness to peripheral stimuli which can be either noxious (hyperalgesia) or non-noxious (allodynia). For example, patients with muscle hyperalgesia will rate painful muscle stimuli higher than normal controls, whereas patients with allodynia may perceive light touch as painful, something that a "normal" individual will never describe as painful. The pathogenesis of such peripheral and/or central nervous system changes in FM is unclear, but peripheral tissue changes, specifically in muscles have been implicated. Indirect evidence from interventions that attenuate tonic peripheral impulse input in patients with FM suggest that overall FM pain is dependent on signals from deep tissues. More importantly, allodynia and hyperalgesia can be improved or abolished by removal of peripheral impulse input. Another potential mechanism for FM pain is central disinhibition. However, this pain mechanism also depends on tonic impulse input even if only inadequately inhibited. Thus a promising approach to understanding FM pain is to determine whether abnormal activity of receptors in deep tissues is fundamental to the development and maintenance of this chronic pain disorder. .COPYRGT. 2007 Bentham Science Publishers Ltd.

L5 ANSWER 9 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2007:22168 BIOSIS
DN PREV200700036860
TI PACAP enhances mouse urinary bladder contractility and is upregulated in micturition reflex pathways after cystitis.
AU Herrera, Gerald M.; Braas, Karen M.; May, Victor; Vizzard, Margaret A.
[Reprint Author]
CS Univ Vermont, Coll Med, Dept Neurol, D411 Given Bldg, Burlington, VT 05405
USA
margaret.vizzard@uvm.edu

SO Vaudry, H [Editor]; Laburthe, M [Editor]. Ann. N. Y. Acad. Sci.,
(2006)
pp. 330-336. Annals of the New York Academy of Sciences.
Publisher: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4
2DQ, OXEN,
UK. Series: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES.
Meeting Info.: 7th International Symposium on VIP, PACAP and
Related
Peptides. Rouen, FRANCE. September 11 -14, 2005. Conseil Reg
Haute-Normandie; Agglomerat Rouen; Inst Fed Rech
Multidisciplinaires
Peptides; Inst Natl Sante Rech Med; Municipal Rouen; Sci Act
Haute-Normandie; Tech Chime-Biol Sante; Univ Paris 7; Univ
Rouen.
CODEN: ANYAA9. ISSN: 0077-8923. ISBN: 1-57331-550-8(H).
DT Book; (Book Chapter)
Conference; (Meeting)
LA English
ED Entered STN: 27 Dec 2006
Last Updated on STN: 11 Jul 2007
AB Pituitary adenylate cyclase-activating polypeptide (PACAP)
elicits a transient contraction, sustained increase in the
amplitude of
spontaneous phasic contractions, and significantly increases the
amplitude
of nerve-mediated contractions in mouse urinary bladder smooth
muscle
(UBSM) strips. PACAP immunoreactivity (IR) is increased in
micturition reflex pathways following cystitis. PACAP may
contribute to altered sensation and bladder overactivity in the
chronic
bladder inflammatory syndrome, interstitial cystitis.

L5 ANSWER 10 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson
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STN
AN 2006:290216 BIOSIS
DN PREV200600295152
TI Botulinum toxin type A inhibits calcitonin gene-
related peptide release from isolated rat bladder.
AU Rapp, David E. [Reprint Author]; Turk, Katherine W.; Bales,
Gregory T.;
Cook, Sean P.
CS Univ Chicago, Pritzker Sch Med, Dept Surg, Urol Sect, 5841 S
Maryland
Ave, MC 6038, Chicago, IL 60637 USA
derapp@yahoo.com
SO Journal of Urology, (MAR 2006) Vol. 175, No. 3, Part 1, pp.
1138-1142.
CODEN: JOURAA. ISSN: 0022-5347.
DT Article
LA English

ED Entered STN: 31 May 2006
Last Updated on STN: 31 May 2006
AB Purpose: Increasing evidence suggests that sensory nerve dysfunction may underlie several urological disorders, including interstitial cystitis and sensory urgency. We determined the effect of botulinum toxin type A (Allergan, Irvine, California) on baseline and chemically evoked release of the sensory neuropeptide, calcitonin gene-related peptide in an isolated bladder preparation. Materials and Methods: Whole rat bladders were incubated in a series of tissue baths containing physiological salt solution. Following bladder equilibration in PSS sequential incubation was performed and this sample was used to measure baseline CGRP release. To evoke CGRP release tissue was subsequently incubated in PSS containing capsaicin (30 nM) and adenosine triphosphate (10 μ M). To measure the effect of BTX-A on baseline and evoked CGRP release bladders were incubated for 6 hours in an organ bath containing BTX-A (50 μ M) or vehicle prior to bladder equilibration. CGRP release was determined by radioimmunoassay. Results: Mean baseline release of CGRP SEM was 346 \pm 44 pg/gm. Adenosine triphosphate/capsaicin application increased CGRP release by 75% over baseline (606 \pm 98 pg/gm, $p < 0.005$). BTX-A application resulted in a 19% decrease in baseline release of CGRP, although this difference did not achieve statistical significance. BTX-A application significantly decreased evoked CGRP by 62% vs control (606 \pm 98 vs 229 \pm 21 pg/gm, $p < 0.005$). Conclusions: BTX-A application inhibits the evoked release of CGRP from afferent nerve terminals in isolated rat bladder. This finding suggests a potential clinical benefit of BTX-A for the treatment of interstitial cystitis or sensory urgency.

L5 ANSWER 11 OF 27 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 4
AN 2006:983267 CAPLUS
DN 145:500253
TI PACAP enhances mouse urinary bladder contractility and is upregulated in micturition reflex pathways after cystitis
AU Herrera, Gerald M.; Braas, Karen M.; May, Victor; Vizzard, Margaret A.
CS Department of Pharmacology, University of Vermont College of Medicine,
Burlington, VT, 05405, USA
SO Annals of the New York Academy of Sciences (2006), 1070 (VIP, PACAP, and

Related Peptides), 330-336
 CODEN: ANYAA9; ISSN: 0077-8923
 PB Blackwell Publishing, Inc.
 DT Journal
 LA English
 AB Pituitary adenylate cyclase-activating polypeptide (PACAP) elicits a transient contraction, sustained increase in the amplitude of spontaneous phasic contractions, and significantly increases the amplitude of nerve-mediated contractions in mouse urinary bladder smooth muscle (UBSM) strips. PACAP immunoreactivity (IR) is increased in micturition reflex pathways following cystitis. PACAP may contribute to altered sensation and bladder overactivity in the chronic bladder inflammatory syndrome, interstitial cystitis.
 OSC.G 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
 RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
 L5 ANSWER 12 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
 AN 2006:221849 BIOSIS
 DN PREV200600225257
 TI Role for pituitary adenylate cyclase activating polypeptide (PACAP) in cystitis-induced plasticity of micturition reflexes.
 AU Braas, K. M. [Reprint Author]; May, V.; Zvara, P.; Nausch, B.; Kliment, J.; Dunleavy, J. D.; Nelson, M.; Vizzard, M. A.
 CS Univ Vermont, Coll Med, Dept Anat and Neurobiol, Burlington, VT 05405 USA
 SO Regulatory Peptides, (SEP 15 2005) Vol. 130, No. 3, pp. 157-158. Meeting Info.: 7th International Symposium on VIP, PACAP and Related Peptides. Rouen, FRANCE. September 11 -14, 2005. Conseil Reg Haute-Normandie; Agglomerat Rouen; Inst Fed Rech Multidisciplinaires Peptides; Inst Natl Sante Rech Med; Municipal Rouen; Sci Act Haute-Normandie; Tech Chime-Biol Sante; Univ Paris 7; Univ Rouen.
 CODEN: REPPDY. ISSN: 0167-0115.
 DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LA English
 ED Entered STN: 5 Apr 2006
 Last Updated on STN: 5 Apr 2006
 L5 ANSWER 13 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

STN
 AN 2005:447062 BIOSIS
 DN PREV200510235533
 TI Innervation induced by cystitis. Comparison of experimental
 cystitis
 modelin pigs versus interstitial cystitis in humans.
 AU Radziszewski, P. [Reprint Author]; Bossowska, A.; Borkowski, A.;
 Majewski,
 M.
 SO European Urology Supplements, (MAR 2005) Vol. 4, No. 3, pp. 58.
 Meeting Info.: 20th Annual Meeting of the
 European-Association-of-Urology.
 Istanbul, TURKEY. 20050317,. European Assoc Urol.
 ISSN: 1569-9056.
 DT Conference; (Meeting)
 Conference; Abstract; (Meeting Abstract)
 LA English
 ED Entered STN: 3 Nov 2005
 Last Updated on STN: 3 Nov 2005

L5 ANSWER 14 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson
 Corporation on
 STN
 AN 2005:8558 BIOSIS
 DN PREV200500004621
 TI Intravesical botulinum toxin A administration produces analgesia
 against
 acetic acid induced bladder pain responses in rats.
 AU Chuang, Yao-Chi [Reprint Author]; Yoshimura, Naoki; Huang,
 Chao-Cheng;
 Chiang, Po-Hui; Chancellor, Michael B.
 CS Suite 700, Kaufmann Bldg, 3471 5th Ave, Pittsburgh, PA, 15213, USA
 chancellormb@msx.upmc.edu
 SO Journal of Urology, (October 2004) Vol. 172, No. 4, Part 1, pp.
 1529-1532.
 print.
 CODEN: JOURAA. ISSN: 0022-5347.
 DT Article
 LA English
 ED Entered STN: 16 Dec 2004
 Last Updated on STN: 16 Dec 2004
 AB Purpose: There is evidence that botulinum toxin A (BTX-A) might
 have
 analgesic properties. However, the mechanisms by which BTX-A
 alters pain
 remain largely unexplored. In the bladder afferent nerve fibers
 contain
 calcitonin gene-related peptide (CGRP). In this study we investigated the effect of intravesical
 BTX-A administration on CGRP immunoreactivity and bladder
 hyperactivity in an acetic acid induced bladder pain model in
 rats.

Materials and Methods: Experimental and control animals were catheterized and intravesically exposed to protamine sulfate (1 ml, 10 mg/ml), followed by BTX-A (1 ml, 25 U/ml) or saline, respectively. Three or 7 days after intravesical therapy continuous cystometrograms were performed using urethane anesthesia by filling the bladder (0.08 ml per minute) with saline, followed by 0.3% acetic acid. Bladder immunohistochemistry was used to detect CGRP. Results: The intercontraction interval (ICI) was decreased after acetic acid instillation (50.2% and 65.0%) in the control group at days 3 and 7, respectively. However, rats that received BTX-A showed a significantly decreased response (28.6% decrease) to acetic acid instillation at day 7. This effect was not observed at day 3 (62.2% ICI decrease). Increased CGRP immunoreactivity was detected in the BTX treated group at day 7, which was not detected at day 3. Conclusions: Intravesical BTX administration blocked acetic acid induced bladder pain responses and inhibited CGRP release from afferent nerve terminals. These results support the clinical application of BTX-A for the treatment of interstitial cystitis and other types of visceral pain.

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 AN 2004315260 EMBASE
 TI Experimental neurogenic cystitis.
 AU Jasmin, Luc (correspondence); Janni, Gabriella
 CS Department of Neurological Surgery, University of California, San Francisco, CA, United States.
 SO Advances in Experimental Medicine and Biology, (2004) Vol. 539
 A, pp. 319-335.
 Refs: 86
 ISSN: 0065-2598 CODEN: AEMBAP
 CY United States
 DT Journal; Conference Article; (Conference paper)
 FS 021 Developmental Biology and Teratology
 028 Urology and Nephrology
 005 General Pathology and Pathological Anatomy
 008 Neurology and Neurosurgery
 009 Surgery

LA English
SL English
ED Entered STN: 12 Aug 2004
Last Updated on STN: 12 Aug 2004
AB Recent advances in basic and clinical research indicate that interstitial cystitis (IC) is a form of neurogenic inflammation, thereby opening new avenues for research into this painful disease. With this in mind, we have recently developed a rat model of neurogenic inflammation of the bladder produced by a central nervous system viral disease. As in IC, the inflammation in this model develops without direct injury or trauma to the bladder, is non-infectious, and is limited to the bladder. Our most recent studies aimed at further testing the similarity of this animal model to IC by assessing the urine content in histamine with the occurrence of mast cell degranulation in the bladder wall. We further verified for a sex difference in the occurrence of the disease. Our results showed increased levels of urine histamine and mast cell activation during the early stages of the disease. We additionally observed that females had a greater degree of plasma extravasation, while males had a greater cellular infiltration together with worse behavioral signs. Gonadectomy prevented the bladder inflammation altogether in both males and females. These findings further validate our model of neurogenic cystitis to study the neurogenic component of IC.

L5 ANSWER 16 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN
AN 2004:256199 BIOSIS
DN PREV200400256219
TI Efficacy and safety of recombinant human anti-NGF antibody in the treatment of IC.
AU Dimitrakov, Jordan D. [Reprint Author]; Dikov, Dorian [Reprint Author]
CS Plovdiv, Bulgaria
SO Journal of Urology, (April 2004) Vol. 171, No. 4 Supplement, pp. 95.
print.
Meeting Info.: Annual Meeting of the American Urological Association. San

Francisco, CA, USA. May 08-13, 2004. American Urological Association.

CODEN: JOURAA. ISSN: 0022-5347.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 12 May 2004

Last Updated on STN: 12 May 2004

L5 ANSWER 17 OF 27 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights

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AN 2003501773 EMBASE

TI Special Contribution 1: The basics behind bladder pain: A review of data

on lower urinary tract sensations.

AU Wyndaele, J.J., Dr. (correspondence); De Wachter, Stefan

CS Department of Urology, Faculty of Medicine, University of Antwerpen,

Belgium. Jean-Jacques.Wyndaele@uza.be

AU Wyndaele, J.J., Dr. (correspondence)

CS Department of Urology, UZA, 10 Wilrijkstraat, B 2650 Edegem, Belgium.

Jean-Jacques.Wyndaele@uza.be

SO International Journal of Urology, (Oct 2003) Vol. 10, No.

SUPPL., pp.

S49-S55.

Refs: 86

ISSN: 0919-8172 CODEN: IJURF3

CY Australia

DT Journal; Conference Article; (Conference paper)

FS 028 Urology and Nephrology

006 Internal Medicine

LA English

SL English

ED Entered STN: 30 Dec 2003

Last Updated on STN: 30 Dec 2003

AB Interstitial cystitis is a syndrome consisting of frequency, urgency, and bladder pain that increases with bladder filling

and improves temporarily after voiding. The exact cause or causes are not

as yet fully understood. This leads to uncertainty in diagnosis and

treatment. There is need for more knowledge, and to acquire this for more

research. The fact that the condition causes pain, a pathologic stimulation of sensory fibres, makes understanding the basic sensory

mechanisms in the lower urinary tract in normal and pathologic conditions

mandatory. In this article we review the data on bladder sensation from

the last 25 years and the possible relation with painful bladder syndrome.

L5 ANSWER 18 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

STN

AN 2002:2909 BIOSIS

DN PREV200200002909

TI Alterations in bladder afferent neurons and urothelium in cats with interstitial cystitis.

AU Buffington, C. A. [Reprint author]; Kiss, S.; Roppolo, J. R.; de Groat, W.

C.; Dineley, K. E.; Reynolds, I. J.; Birdier, L. A.

CS College Veterinary Medicine, Ohio State University, Columbus, OH, USA

SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 2163.

print.

Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San

Diego, California, USA. November 10-15, 2001.

ISSN: 0190-5295.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 28 Dec 2001

Last Updated on STN: 25 Feb 2002

AB Experiments were conducted in cats with feline interstitial cystitis (IC) to evaluate whether the chemical properties and/or intracellular signaling mechanisms in afferent neurons and epithelial

cells in the urinary bladder (UB) are altered in IC. UB dorsal root

ganglion (DRG) cells were identified by axonal tracing (fast blue) and

sections of DRG, spinal cord (SC) and UB were processed for neuropeptides

(CGRP, VIP). In IC cats, the number of CGRP

-immunoreactive dye-labelled, bladder DRG cells was increased by 50% and

the mean size of labelled DRG cells was increased (45%).

Afferent (VIP,

CGRP) fiber density in UB and sacral spinal cord also increased in

IC. In addition, epithelial cells in IC cats exhibited abnormal calcium

signaling. In urothelial cells from normal cat UB, ATP mobilized intracellular calcium via activation of P2Y receptors, whereas both P2X

and P2Y receptors were involved in this response in cells from IC cats.

In addition, compared to normal cats, cultured urothelial cells from IC cats exhibited a significant increase (90%) in stretch-evoked ATP release induced by a hypo-osmotic stimulus measured using a luciferin-luciferase assay. ATP release was blocked by gadolinium, an inhibitor of stretch activated channels. These studies revealed that IC cats have an altered urothelium which in turn may influence afferent excitability. Changes in neural-epithelial interactions may correlate with abnormal sensations in IC.

L5 ANSWER 19 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

STN

DUPLICATE 6

AN 2001:345507 BIOSIS

DN PREV200100345507

TI Cell relationship in a wistar rat model of spontaneous prostatitis.

AU Keith, Ingegerd M. [Reprint author]; Jin, Jie; Neal, Durwood, Jr.;

Teunissen, Brian D.; Moon, Timothy D.

CS Departments of Comparative Bioscience and Surgery, University of Wisconsin

and Veterans Affairs Medical Center, Madison, WI, USA

SO Journal of Urology, (July, 2001) Vol. 166, No. 1, pp. 323-328. print.

CODEN: JOURAA. ISSN: 0022-5347.

DT Article

LA English

ED Entered STN: 25 Jul 2001

Last Updated on STN: 19 Feb 2002

AB Purpose: Prostatitis in men is a painful, noninfectious inflammatory

condition. It is similar to interstitial cystitis which is associated with increased bladder mast cell and sensory nerve

fiber density as well as suprapubic pain. Certain strains of rats may

provide a useful model for studies of the development of spontaneous

prostatitis. We evaluated the time course, and involvement of mast cells

and sensory nerve fibers in this process using Wistar rats.

Materials and

Methods: The prostates of 4, 6, 8, 10 and 13-week-old male Wistar rats

were examined for the degree of inflammation, innervation, mast cell

density and nerve mast cell relationship using histochemical and immunocytochemical studies. Bacterial cultures of tissue were performed at 13 weeks. Results: The inflammatory cell index increased progressively with age. Inflammation was moderate and consisted mostly of lymphocytes and macrophages associated with occasional glandular epithelial necrosis and edema. The density of nerve fibers immunoreacting with the neuronal marker protein gene produce 9.5 increased gradually with age and fibers immunopositive for the sensory neuropeptide calcitonin gene-related peptide more than doubled by 13 weeks compared with by 4 weeks. The density of visible mast cells declined after 4 weeks in a pattern that corresponded with the increased percent of mast cells undergoing degranulation. For the mast cells with calcitonin gene-related peptide immuno-positive nerve fibers within a distance of 40 μ m. distance correlated significantly with the degree of degranulation. Bacterial cultures were negative at 13 weeks. Conclusions: Our results confirm previous reports of spontaneous prostatitis in Wistar rats and indicate that moderate inflammation may occur in 80% of rats at as early as age 13 weeks. While the correlation of the nerve mast cell axis with mast cell degranulation does not prove our hypothesis of mast cell mediated inflammatory mediator release in the development of nonbacterial prostatitis, it suggests that such a relationship is possible.

L5 ANSWER 20 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 7
 AN 2001:284434 BIOSIS
 DN PREV200100284434
 TI Alterations in neuropeptide expression in lumbosacral bladder pathways following chronic cystitis.
 AU Vizzard, Margaret A. [Reprint author]
 CS Department of Anatomy and Neurology, University of Vermont College of Medicine, E219 Given Building, Burlington, VT, 05405, USA
 mvizzard@zoo.uvm.edu
 SO Journal of Chemical Neuroanatomy, (March, 2001) Vol. 21, No. 2, pp.

125-138. print.

CODEN: JCNAEE. ISSN: 0891-0618.

DT Article

LA English

ED Entered STN: 13 Jun 2001

Last Updated on STN: 19 Feb 2002

AB These studies examined changes in the expression of calcitonin gene-related peptide (CGRP) and substance P (SP) in lumbosacral (L6-S1) micturition reflex pathways,

following chronic cystitis induced by cyclophosphamide (CYP).

In control

Wistar rats, CGRP- or SP-immunoreactivity (IR) was expressed in fibers in the superficial dorsal horn in all segmental levels examined

(L4-S1). Bladder afferent cells in the dorsal root ganglia (DRG; L6, S1)

from control animals also exhibited CGRP-(41-55%) or SP-IR (2-3%). Following chronic, CYP-induced cystitis, CGRP- and SP-IR were dramatically increased in spinal segments and DRG

(L6, S1) involved in micturition reflexes. The density of CGRP- and SP-IR was increased in the superficial laminae (I-II) of the L6 and S1

spinal segments. No changes in CGRP- or SP-IR were observed in the L4-L5 segments. Staining was also dramatically increased in a fiber

bundle extending ventrally from Lissauer's tract in lamina I along the

lateral edge of the DH to the sacral parasympathetic nucleus in the L6-S1

spinal segments. Following chronic cystitis, CGRP- and SP-IR in cells in the L6 and S1 DRG significantly (P ltoreq 0.05)

increased and the percentage of bladder afferent cells expressing CGRP- (76%) or SP-IR (11-18%) also significantly (P ltoreq 0.001) increased.

No changes

were observed in the L4-L5 DRG. These studies suggest that the neuropeptides, CGRP and SP, may play a role in urinary bladder afferent pathways, following chronic urinary bladder

inflammation.

Changes in CGRP or SP expression following cystitis may contribute to the altered visceral sensation (allodynia) and/or urinary

bladder hyperreflexia in the clinical syndrome, interstitial cystitis.

L5 ANSWER 21 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

STN

DUPLICATE 8

AN 2000:172201 BIOSIS

DN PREV200000172201

TI Increased tyrosine hydroxylase immunoreactivity in bladder tissue from patients with classic and nonulcer interstitial cystitis

AU Peeker, Ralph [Reprint author]; Aldenborg, Frank; Dahlstrom, Annica;
Johansson, Sonny L.; Li, Jia-Yi; Fall, Magnus

CS Urology Division, Department of Surgery, Sahlgrenska University Hospital,
Goteborg, Sweden

SO Journal of Urology, (April, 2000) Vol. 163, No. 4, pp. 1112-1115. print.
CODEN: JOURAA. ISSN: 0022-5347.

DT Article

LA English

ED Entered STN: 3 May 2000
Last Updated on STN: 4 Jan 2002

AB Purpose: Interstitial cystitis is a chronic debilitating condition which mainly affects women. Accumulated evidence indicates that interstitial cystitis is a heterogeneous syndrome. The nonulcer subtype appears different than classic interstitial cystitis in regard to symptoms, and endoscopic and histological findings as well as response to various treatments. We further explore the neurogenic nature of this disease using indirect immunofluorescence to evaluate the presence and density of various autonomic and sensory nerve fibers. Materials and Methods: Specimens from the bladder wall of 6 patients with classic interstitial cystitis, 7 with nonulcer interstitial cystitis and 6 controls were evaluated to determine the presence and density of nerve fibers containing tyrosine hydroxylase, calcitonin gene-related peptide, neuropeptide Y and substance P using specific antibodies, and the general presence of nerve fibers using a mixture of antibodies against nerve filament, neuron specific enolase and S-100 protein. Results: Increased density and number of nerve fibers immunoreactive for tyrosine hydroxylase were noted in interstitial cystitis cases compared to controls. Furthermore, there was a difference between classic and nonulcer disease in the overall density of nerves using the antibody mixture. Conclusions: Our findings indicate an altered

peripheral sympathetic innervation in interstitial cystitis cases, which may be an indication of primary neurogenic etiology. The difference in nerve density observed after incubation with the antibody mixture between classic and nonulcer interstitial cystitis supports the hypothesis that the 2 forms represent separate entities.

L5 ANSWER 22 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 9

AN 2000:221490 BIOSIS

DN PREV200000221490

TI Up-regulation of pituitary adenylate cyclase-activating polypeptide in

urinary bladder pathways after chronic cystitis.

AU Vizzard, Margaret A. [Reprint author]

CS College of Medicine, Department of Neurology, University of Vermont, E219

Given Building, Burlington, VT, 05405, USA

SO Journal of Comparative Neurology, (May 8, 2000) Vol. 420, No. 3, pp.

335-348. print.

CODEN: JCNEAM. ISSN: 0021-9967.

DT Article

LA English

ED Entered STN: 31 May 2000

Last Updated on STN: 5 Jan 2002

AB These studies examined changes in the expression of pituitary adenylate

cyclase-activating polypeptide (PACAP) in micturition reflex pathways after chronic cystitis induced by cyclophosphamide (CYP). In

control Wistar rats, PACAP immunoreactivity was expressed in fibers in the superficial dorsal horn at all segmental levels examined

(L1, L2, and L4-S1). Bladder afferent cells (40-45%) in the dorsal root

ganglia (DRG; L1, L2, L6, and S1) from control animals also exhibited

PACAP immunoreactivity. After chronic, CYP-induced cystitis, PACAP immunoreactivity increased dramatically in spinal segments and DRG (L1, L2, L6, and S1) involved in micturition reflexes.

The

density of PACAP immunoreactivity was increased in the superficial laminae (I-II) of the L1, L2, L6, and S1 spinal segments. No

changes in PACAP immunoreactivity were observed in the L4-L5 segments. Staining also increased dramatically in a fiber bundle extending ventrally from Lissauer's tract in lamina I along the lateral

edge of the dorsal horn to the sacral parasympathetic nucleus in the L6-S1

spinal segments (lateral collateral pathway of Lissauer). After chronic cystitis, PACAP immunoreactivity in cells in the L1, L2, L6, and S1 DRG increased significantly ($P < 0.0001$), and the percentage of bladder afferent cells expressing PACAP immunoreactivity also increased significantly ($P < 0.0001$; 70-85%). No changes were observed in the L3-L5 DRG. These studies suggest that the neuropeptide, PACAP, may play a role in urinary bladder afferent pathways after visceral (urinary bladder) inflammation. Changes in PACAP expression after cystitis may play a role in altered visceral sensation (allodynia) and/or urinary bladder hyperreflexia in the clinical syndrome, interstitial cystitis.

L5 ANSWER 23 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

STN
AN 2001:96859 BIOSIS
DN PREV200100096859
TI Alterations in urothelium and bladder afferents in feline interstitial cystitis.
AU Buffington, C. A. [Reprint author]; Kiss, S.; Kanai, A. J.; Dineley, K.; Roppolo, J. R.; Reynolds, I. J.; de Groat, W. C.; Birdner, L. A.
CS Ohio State University, Columbus, OH, USA
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-349.2. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 21 Feb 2001
Last Updated on STN: 15 Feb 2002
AB The properties of bladder afferent neurons and urothelial cells were examined in normal cats and in cats diagnosed with FIC, a chronic painful disorder of the urinary bladder (UB). The UB, sacral dorsal root ganglia (DRG) and spinal cord (SC), were removed from anesthetized adult cats of either sex before or after perfusion fixation. Small numbers of c-jun-immunoreactive bladder DRG cells were detected in normal cats (<4

cells/section), but the numbers increased (200%) in cats with FIC. UB-DRG cells, labeled by axonal tracers were larger (25%) in FIC cats. The density of substance P and CGRP containing afferent nerves in the UB and spinal dorsal horn was greater in FIC cats. Basal nitric oxide (NO) release, measured with a microsensor in bladder strips, was detected in FIC cats but not in normal cats, whereas NO release evoked by capsaicin was decreased (60%) in normal cats. The UB of FIC cats displayed regions of denuded uroepithelium as evidenced by changes in cytokeratin staining. In cultured uroepithelial cells intracellular calcium measurements using Fura-2 and fluorescent microscopic techniques revealed that sensitivity to purinergic agents was altered in FIC cats. Activation of P2Y receptors (2-methylthio ATP) increased calcium in normal cats, whereas activation of P2X (alpha,beta methylene ATP) or P2Y receptors was effective in FIC cats. These studies raise the possibility that changes in properties of afferent nerves and/or the urothelium may contribute to the painful symptoms in FIC.

L5 ANSWER 24 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN
AN 1999:160213 BIOSIS
DN PREV199900160213
TI Autonomous neuropathy in interstitial cystitis.
AU Peeker, Ralph [Reprint author]; Aldenborg, Frank [Reprint author]; Li, Jia-Yi [Reprint author]; Fall, Magnus [Reprint author]; Dahlstrom, Annica [Reprint author]; Johansson, Sonny L.
CS Gothenburg, Sweden
SO Journal of Urology, (April, 1999) Vol. 161, No. 4 SUPPL., pp. 28. print.
Meeting Info.: 94th Annual Meeting of the American Urological Association, Inc. Dallas, Texas, USA. May 1-6, 1999. American Urological Association.
CODEN: JOURAA. ISSN: 0022-5347.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English
ED Entered STN: 16 Apr 1999
Last Updated on STN: 16 Apr 1999

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AN 1997291246 EMBASE

TI Neurophysiology of micturition and continence in women.

AU Chai, T.C.; Steers, W.D., Prof. (correspondence)

CS University of Virginia Health Sciences Center, Department of Urology,

Charlottesville, VA, United States.

AU Steers, W.D., Prof. (correspondence)

CS University of Virginia Health Sciences Center, Department of Urology, Box

422, Charlottesville, VA 22908, United States.

AU Steers, W.D., Prof. (correspondence)

CS Univ. Virginia Health Sciences Ctr., Department of Urology, Box 422,

Charlottesville, VA 22908, United States.

SO International Urogynecology Journal and Pelvic Floor Dysfunction, (1997)

Vol. 8, No. 2, pp. 85-97.

Refs: 150

ISSN: 0937-3462 CODEN: IUFDV

CY United Kingdom

DT Journal; General Review; (Review)

FS 010 Obstetrics and Gynecology

028 Urology and Nephrology

030 Clinical and Experimental Pharmacology

037 Drug Literature Index

008 Neurology and Neurosurgery

LA English

SL English

ED Entered STN: 9 Oct 1997

Last Updated on STN: 9 Oct 1997

AB Micturition and continence involve the coordination of complex neural

events between the central and peripheral nervous systems. An understanding of these events provides a foundation for the treatment of voiding disorders in women such as stress urinary incontinence, urge

incontinence and interstitial cystitis. The purpose of this paper is to comprehensively review the neuroanatomy, neurophysiology and neuropharmacology of micturition and continence.

However, a brief section discussing clinical correlations will follow each

of these topics to help integrate the basic science with clinical observations.

L5 ANSWER 26 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 10

AN 1992:218126 BIOSIS

DN PREV199293118351; BA93:118351

TI INTERSTITIAL CYSTITIS INCREASED SYMPATHETIC INNERVATION AND RELATED NEUROPEPTIDE SYNTHESIS.

AU HOHENFELLNER M [Reprint author]; NUNES L; SCHMIDT R A; LAMPEL A; THUEROFF J W; TANAGHO E A

CS DEP UROL, KLINIKUM BARMEN, HEUSNERSTR 40, 5600 WUPPERTAL, WEST GERMANY

SO Journal of Urology, (1992) Vol. 147, No. 3 PART 1, pp. 587-591. CODEN: JOURAA. ISSN: 0022-5347.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 4 May 1992
Last Updated on STN: 5 May 1992

AB To investigate the possibility of a neural deterioration of the bladder wall in interstitial cystitis, bladder tissue from 10 patients with interstitial cystitis was compared with that from 10 control subjects by means of immunohistochemistry.

An enhanced innervation of the bladder in the submucosa and detrusor muscle was found to represent an increase of sympathetic but not cholinergic neurons. In interstitial cystitis the number of neurons positive for vasoactive intestinal polypeptide and neuropeptide Y was higher and carried a larger number of axonal varicosities, whereas the number of neurons positive for substance P and calcitonin-gene-related peptide was not significantly different in both groups. We conclude that interstitial cystitis is associated with increased sympathetic outflow into the bladder and altered metabolism of vasoactive intestinal polypeptide and neuropeptide Y. Since similar changes have been observed in other inflammatory diseases of a presumably autoimmune nature, such as rheumatoid arthritis, Crohn's disease and colitis ulcerosa, the pathophysiology of interstitial cystitis may share common pathways with the latter. Experience in these diseases may facilitate a better understanding of the pathophysiology of interstitial cystitis and suggest new therapeutic concepts.

L5 ANSWER 27 OF 27 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
 STN
 AN 1992:298493 BIOSIS
 DN PREV199243010843; BR43:10843
 TI IMMUNOHISTOCHEMICAL EXAMINATION OF NEUROPEPTIDES AND M-2 MUSCARINIC
 RECEPTORS IN NORMAL AND INTERSTITIAL CYSTITIS IC BLADDERS.
 AU SHICKLEY T J [Reprint author]; LUTHIN G F; RUGGIERI M R
 CS PHILADELPHIA, PA, USA
 SO Journal of Urology, (1992) Vol. 147, No. 4 SUPPL, pp. 462A.
 Meeting Info.: AUA (AMERICAN UROLOGICAL ASSOCIATION)
 EIGHTY-SEVENTH ANNUAL
 MEETING, WASHINGTON, D.C., USA, MAY 10-14, 1992. J UROL.
 CODEN: JOURAA. ISSN: 0022-5347.
 DT Conference; (Meeting)
 FS BR
 LA ENGLISH
 ED Entered STN: 16 Jun 1992
 Last Updated on STN: 16 Jun 1992

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=> s interstitial cystitis
L6 4413 INTERSTITIAL CYSTITIS

=> d his

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FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:15:16 ON 25 JAN 2010
L1 31928 S CALCITONIN GENE RELATED PEPTIDE OR CGRP
L2 9041 S PITUITARY ADENYLATE CYCLASE ACTIVATING PEPTIDE OR
PACAP
L3 40576 S L1 OR L2
L4 43 S L3 AND INTERSTITIAL CYSTITIS
L5 27 DUP REM L4 (16 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 17:23:30 ON 25 JAN 2010

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:35:03 ON 25 JAN 2010
L6 4413 S INTERSTITIAL CYSTITIS

=> s l6 and diagnos?
L7 1735 L6 AND DIAGNOS?

=> s l7 and review
L8 316 L7 AND REVIEW

=> d bib abs 1-10

L8 ANSWER 1 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson
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STN
AN 2010:6079 BIOSIS
DN PREV201000006079
TI Bladder Outlet Obstruction in Painful Bladder Syndrome/
Interstitial Cystitis.
AU Cameron, Anne P. [Reprint Author]; Gajewski, Jerzy B.
CS 1500 Med Ctr Dr,3875 Taubman Ctr, Ann Arbor, MI 48109 USA
annepell@med.umich.edu
SO Neurourology and Urodynamics, (2009) Vol. 28, No. 8, pp.
944-948.
CODEN: NEUREM. ISSN: 0733-2467.
DT Article

LA English
ED Entered STN: 9 Dec 2009
Last Updated on STN: 9 Dec 2009
AB Aims: Obstructive symptoms such as slow stream, dribbling and straining are often reported by painful bladder syndrome and interstitial cystitis (PBS/IC) patients. Our hypothesis was that some patients with PBS/IC have an associated measurable bladder outlet obstruction (BOO) secondary to dysfunctional voiding and that those patients with more severe PBS/IC are more likely to have BOO. Methods: This is a retrospective chart review of female patients diagnosed with PBS/IC based on the NIDDK research definition. Charts were reviewed for clinical symptom severity, ulcer or non-ulcer PBS/IC on cystoscopy, and pressure-flow urodynamics (UDPF). Patients were excluded if they had a urinary infection at the time of urodynamics or did not meet study entry requirements. The cut-off values of ≤ 12 ml/sec and ≥ 25 cm of water was used to define BOO. Results: Of the 231 women: 38 had ulcer PBS/IC and 193 had non-ulcer PBS/IC. MCC was 269 ml in non-ulcer PBS/IC and 200 ml in ulcer PBS/IC ($P = 0.006$). One hundred eleven women (48%) met criteria for obstruction. MCC was 298 ml in the non-obstructed group and 214 ml in the obstructed group ($P < 0.0001$). The maximum flow with non-ulcer PBS/IC was 11.0 ml/sec and in ulcer PBS/IC 8.9 ml/sec ($P = 0.04$). Detrusor pressure at maximum flow was 33.3 cm H₂O, in non-ulcer, and 37.4 cm H₂O in ulcer PBS/IC ($P = 0.01$). Conclusions: Forty-eight percent of our PBS/IC patients have BOO, and increasing severity of PBS/IC is associated with higher voiding pressure. Neurourol. Urodynam. 28:944-948, 2009. (C) 2009 Wiley-Liss, Inc.

L8 ANSWER 2 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

STN
AN 2009:298223 BIOSIS
DN PREV200900299326
TI Developmental Influences on Medically Unexplained Symptoms.
AU Buffington, C. A. Tony [Reprint Author]

CS Ohio State Univ, Vet Hosp, 601 Tharp St, Columbus, OH 43210 USA
Buffington.1@osu.edu

SO Psychotherapy and Psychosomatics, (2009) Vol. 78, No. 3, pp.
139-144.
CODEN: PSPSBF. ISSN: 0033-3190.

DT Article
General Review; (Literature Review)

LA English

ED Entered STN: 6 May 2009
Last Updated on STN: 20 May 2009

AB Background: Medically unexplained (or 'functional') symptoms
(MUS) are
physical symptoms that prompt the sufferer to seek healthcare
but remain
unexplained after an appropriate medical evaluation. Examples
of MUS also
occur in veterinary medicine. For example, domestic cats suffer
a
syndrome comparable to interstitial cystitis, a
chronic pelvic pain syndrome of humans. Method: Review of
current evidence suggests the hypothesis that developmental
factors may
play a role in some cases of MUS. Maternal perception of a
threatening
environment may be transmitted to the fetus when hormones cross
the
placenta and affect fetal physiology, effectively 'programming'
the fetal
stress response system and associated behaviors toward enhanced
vigilance.
After birth, intense stress responses in the individual may
result in
similar vulnerability, which may be unmasked by subsequent
stressors.
Results: Epigenetic modulation of gene expression (EMGEX)
appears to play
a central role in creation of this 'survival phenotype'. The
recent
development of techniques to identify the presence of EMGEX
provides new
tools to investigate these questions, and drugs and other
interventions
that may reverse EMGEX are also under active investigation.
Conclusion:
Viewing MUS from the perspective of underlying developmental
influences
involving EMGEX that affect function of a variety of organs
based on
familial (genetic and environmental) predispositions rather than
from the
traditional viewpoint of isolated organoriginating diseases has
at least

two important implications: it provides a parsimonious explanation for findings heretofore difficult to reconcile, and it opens whole new areas of investigation into causes and treatments for this class of disorders.

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L8 ANSWER 3 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

AN 2009:185242 BIOSIS

DN PREV200900185242

TI Breaking the Cycle of Pain in Interstitial Cystitis /Painful Bladder Syndrome Toward Standardization of Early Diagnosis and Treatment.

AU Forrest, John B. [Reprint Author]; Mishell, Daniel R. Jr.

CS 10901 E 48th St S, Tulsa, OK 74146 USA

jforrest@sjmc.org

SO Journal of Reproductive Medicine, (JAN 2009) Vol. 54, No. 1, pp. 3-14.

CODEN: JRPMAP. ISSN: 0024-7758.

DT Article

General Review; (Literature Review)

LA English

ED Entered STN: 11 Mar 2009

Last Updated on STN: 11 Mar 2009

AB Chronic pelvic pain (CPP) affects about 15% of female adults in the United

States. The source of this pain in many women is the bladder, specifically interstitial cystitis/painful bladder syndrome (IC/PBS). Despite the frequent occurrence of IC/PBS as

a cause

of CPP, there currently are no universally accepted guidelines

for

diagnosis and treatment of this disorder, and, consequently, many patients do not receive appropriate treatment in a timely

manner. In an

effort to develop a rational way to diagnose and treat patients

With CPP, a panel of leaders in urology, gynecology,

urogynecology and

general women's health met to review recent literature, reach consensus and formulate 2 algorithms, one for diagnosing and the other for managing IC/PBS. This article reflects the results of

that

meeting. (J Reprod Med 2009;54:3-14)

L8 ANSWER 4 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

AN 2009:162050 BIOSIS

DN PREV200900162050

TI The Spectrum of Eosinophilic Cystitis in Males Case Series and Literature Review.
 AU Popescu, Oana-Eugenia; Landas, Steve K. [Reprint Author]; Haas, Gabriel P.
 CS State Univ New York Upstate Med Univ, Dept Pathol and Urol, 750 E Adams St, Syracuse, NY 13210 USA
 landas@upstate.edu
 SO Archives of Pathology & Laboratory Medicine, (FEB 2009) Vol. 133, No. 2, pp. 289-294.
 CODEN: APLMAS. ISSN: 0003-9985.
 DT Article
 LA English
 ED Entered STN: 4 Mar 2009
 Last Updated on STN: 4 Mar 2009
 AB Context.-Eosinophilic cystitis (EC) is an inflammatory condition of the bladder that has been linked to food allergens, infectious agents, drugs, and other genitourinary conditions. Like interstitial cystitis, EC has a strong female predominance. It is characterized by an intense eosinophilic infiltrate in the acute phase and fibrosis in the chronic phase.Objectives.-To document and focus on specific features of EC in males and highlight the relationship between clinical and histopathologic findings.Design.-The bladder biopsies of male patients were reviewed. Eight cases of EC were selected.Results.-Several known associations were noted as well as unreported features and associations such as Charcot-Leyden crystals, celiac disease, lupus anticoagulant, and additional viral and bacterial agents.Conclusions.-Eosinophilic cystitis represents a response to a variety of agents and may often be overlooked. The temporally biphasic morphologic features are the hallmark of this condition. Because clinical and imaging studies are not specific, a high index of clinical suspicion is often crucial to the correct diagnosis and proper management of EC.

L8 ANSWER 5 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
 AN 2008:538440 BIOSIS

DN PREV200800538439
 TI Disorders of adhesions or adhesion-related disorder: Monolithic entities
 or part of something bigger - CAPPS?.

AU Wiseman, David M. [Reprint Author]
 CS Int Adhes Society, PMB 238,6757 Arapaho,Suite 711-238, Dallas, TX 75248
 USA
 david.wiseman@adhesions.org

SO Seminars in Reproductive Medicine, (JUL 2008) Vol. 26, No. 4, pp. 356-368.
 ISSN: 1526-8004.

DT Article
 LA English
 ED Entered STN: 1 Oct 2008
 Last Updated on STN: 1 Oct 2008

AB The purpose of this article is to review progress in the field of abdominopelvic adhesions and the validity of its two underlying assumptions: (1) The formation of adhesions results in infertility, bowel obstruction, or other complications. Reducing or avoiding adhesions will curb these sequelae. (2) "Adhesions" is a monolithic entity to be tackled without regard to any other condition. Evidence is discussed to validate the first assumption. We reviewed progress in the field by examining hospital data. We found a growing trend in the number and cost of discharges for just two adhesion-related diagnoses, and the low usage of adhesion barriers appears in at most 5% of appropriate procedures. Data from an Internet-based survey suggested that the problem may, be partly due to ignorance among patients and physicians about adhesions and their prevention. Two other surveys of patients visiting the adhesions.org Web site defined more fully adhesion-related disorder (ARD). The first survey (N= 466) described a patient with chronic pain, gastrointestinal disturbances, an average of nine bowel obstructions, and an inability to work or maintain family or social relationships. The second survey (687 U.S. women) found a high (co-) prevalence of abdominal or pelvic adhesions (85%), chronic abdominal or pelvic pain (69%), irritable bowel syndrome (55%), recurrent bowel obstruction (44%),

endometriosis (40%), and interstitial cystitis (29%). This pattern suggests that although "adhesions" may, start out as a monolithic entity, an adhesions patient may develop related conditions (ARD) until they merge into an independent entity where they are practically indistinguishable from patients with multiple symptoms originating from other abdominopelvic conditions such as pelvic or bladder pain. Rather than use terms that constrain the required multidisciplinary, biopsychosocial approach to these patients by the paradigms of the specialty related to the patient's initial symptom set, the term complex abdominopelvic and pain syndrome (CAPPS) is proposed. It is essential to understand not only the pathogenesis of the "initiating" conditions but also how they progress to CAPPS. In our ARD sample, not only was the frequency of women with hysterectomies (56%) higher than expected (21 to 33%), but also the rates of the "initiating" conditions was 40 to 400% higher in patients with hysterectomies than in those without. This may represent increased surgical trauma or the loss of protection against oxidative stress. Related was the higher frequency of ARD patients reporting hemochromatosis (HC; 5%) than expected (similar to 0.5%) and the higher rates (20 to 700%) of initiating conditions in patients with HC than in those without HC. Together with findings related to the toxicity of InterGel, these findings raise the possibility, that heterozygotes for genes regulating oxidative stress are at greater risk of developing surgical complications as well as more severe and progressive conditions such as CAPPS.

L8 ANSWER 6 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on

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AN 2008:395348 BIOSIS

DN PREV200800395347

TI Urinary tract infection and inflammation at onset of interstitial cystitis/painful bladder syndrome.

AU Warren, John W. [Reprint Author]; Brown, Vivian; Jacobs, Stephen; Horne, Linda; Langenberg, Patricia; Greenberg, Patty

CS Univ Maryland, Sch Med, Dept Med, 10 S Pine St, Room 9-00 MSTF, Baltimore, MD 21201 USA
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SO Urology, (JUN 2008) Vol. 71, No. 6, pp. 1085-1090.
ISSN: 0090-4295.

DT Article

LA English

ED Entered STN: 16 Jul 2008
Last Updated on STN: 16 Jul 2008

AB OBJECTIVES Interstitial cystitis/painful bladder syndrome (IC/PBS) is a chronic disease primarily in women that is of low incidence and, unknown etiology and manifests as bladder pain and urinary symptoms. Acute urinary tract infection (UTI) is of high incidence in women, presents as dysuria and urinary symptoms, and is caused by uropathogenic bacteria. We hypothesized that UTI is present at the onset of IC/PBS in some women. METHODS For a case-control study seeking risk factors for IC/PBS, women with IC/PBS symptoms of 12 months or less were recruited and evaluated by interview and medical record review. The date of symptom onset was identified by a six-step process. Three evidence-based methods using culture, urinalysis, and symptoms were used separately and in combination to diagnose UTI at IC/PBS onset. RESULTS Of 1177 screened women, 314 with recent-onset IC/PBS, including numerous confirming characteristics, were enrolled in the study; 98% of the requested medical records were obtained and reviewed. Evidence of a UTI at the onset of IC/PBS was found in 18% to 36% of women. Common UTI features not used in its diagnosis (short interval to medical care, hematuria, antibiotic treatment, and improvement after antibiotics) were significantly more common in those with onset UTI than in those without. CONCLUSIONS These retrospective data suggest that a proportion, probably a minority, of women at IC/PBS onset had evidence of UTI or inflammation. Our results indicate that UTI is present at the

onset of IC/PBS in some women and might reveal clues to IC/PBS pathogenesis.

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AN 2008:304655 BIOSIS

DN PREV200800306707

TI Characterization of a clinical cohort of 87 women with interstitial cystitis/painful bladder syndrome.

AU Peters, Kenneth M.; Carrico, Donna J. [Reprint Author]; Diokno, Ananias C.

CS William Beaumont Hosp, Dept Urol, Ministrelli Program Urol Res and Educ,

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SO Urology, (APR 2008) Vol. 71, No. 4, pp. 634-640.

ISSN: 0090-4295.

DT Article

LA English

ED Entered STN: 12 May 2008

Last Updated on STN: 12 May 2008

AB OBJECTIVE To provide a characterization of a cohort of women with interstitial cystitis/painful bladder syndrome (IC/PBS) by describing their historical and clinical characteristics.

This was

reported with the National Institutes of Health chronic

prostatitis

cohort, but a literature review did not reveal a similar study

for women with IC/PBS. METHODS A total of 87 women with IC/PBS

were

referred to the Beaumont Women's Initiative for Pelvic Pain and Sexual

Health program. A certified nurse practitioner took a comprehensive

history and per-formed a pelvic exam for each. Data were

analyzed using

descriptive statistics to describe this cohort. RESULTS Most women experienced constant pain for 5 or more years (mean Visual

Analogue Scale =

5 out of 10). A total of 94.2% had levator pain. More than 50%

had

vulvar pain with exam. More than half reported a history of abuse, often

in more than one life stage. A total of 28% had cesarean births and 76%

had a history of miscarriage, stillbirth, or abortion. Women averaged 4

lifetime pelvic surgeries, and 48% had hysterectomies, two-thirds of which

were done before IC/PBS diagnosis. Premenstrual women reported pain throughout the menstrual cycle. As many as 12% had

chlamydia

previously, which was higher than the national average. Common comorbidities were pelvic pain (93%), allergies (86%), and sexual dysfunction (72%).CONCLUSIONS This population of women with unrelieved

chronic pain, frequency, and urgency is in desperate need of care.

Researchers should continue to search for the etiology, prevention, and treatment interventions that are effective in dealing with IC/PBS. It may

be most therapeutic to develop a multimodal plan of care that includes

physical therapy, oral and intravesical therapies, neuromodulation, and cognitive-behavioral therapies.

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STN

AN 2008:139703 BIOSIS

DN PREV200800138354

TI Reprogramming requirements after sacral nerve stimulator implantation:

Correlation with preoperative indication.

AU Maxwell, Kelly M.; Clemens, J. Quentin; Mazzenga, Laura; Kielb, Stephanie

J. [Reprint Author]

CS Northwestern Univ, Feinberg Sch Med, Dept Urol, 303 E Chicago Ave, Tarry

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SO Journal of Urology, (FEB 2008) Vol. 179, No. 2, pp. 549-551.

CODEN: JOURAA. ISSN: 0022-5347.

DT Article

LA English

ED Entered STN: 20 Feb 2008

Last Updated on STN: 20 Feb 2008

AB Purpose: Recent publications support sacral nerve stimulator implantation

in patients with interstitial cystitis. To our

knowledge the reprogramming requirements for all patients

following

stimulator implantation has not been described and it is unknown

whether

the number of sessions required vary by pre-implantation

diagnosis

. We determined overall reprogramming requirements following

nerve

stimulator implantation and whether requirements vary based on

preoperative indication. Materials and Methods: After obtaining

institutional review board approval we retrospectively reviewed

the records of all patients who underwent sacral nerve stimulator

implantation at our institution between June 2002 and October 2004. The preoperative indication and number of reprogramming sessions during the initial test period (stage 1) and following permanent implantation (stage 2) were compared. Results: The 17 patients proceeding to stage 2 with a minimum 12-month followup during the study period were included. Mean age was 43 years (range 26 to 78) and all patients except 1 were female. Patients were separated by diagnosis for evaluation purposes, including urgency/frequency/incontinence in 8, urinary retention in 2 and interstitial cystitis in 7. The average number of reprogramming sessions during stage 1 was 0.9, 3.5 and 2.3 for urgency/frequency/incontinence, urinary retention and interstitial cystitis, respectively. The average number of reprogramming sessions after stage 2 was 2.8, 3.0 and 6.9 at 12-month followup for urgency/frequency/incontinence, urinary retention and interstitial cystitis, respectively. No patient had the stimulator removed for reprogramming failure. Conclusions: Patients in urinary retention appear to require more frequent reprogramming during stage 1, while patients with interstitial cystitis require more sessions after stage 2 implantation.

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STN

AN 2008:85285 BIOSIS

DN PREV200800078702

TI Pharmacologic management of painful bladder syndrome/interstitial cystitis: A systematic review (vol 167, pg 1922, 2007).

AU Dimitrakov, J.; Kroenke, K.; Steers, W. D.; Berde, C.;

Zurakowski, D.;

Freeman, M. R.; Jackson, J. L.

SO Archives of Internal Medicine, (DEC 10 2007) Vol. 167, No. 22, pp. 2452.

CODEN: AIMDAP. ISSN: 0003-9926.

DT Article

Errata

LA English

ED Entered STN: 23 Jan 2008

Last Updated on STN: 23 Jan 2008

L8 ANSWER 10 OF 316 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on
STN
AN 2008:35914 BIOSIS
DN PREV200800035088
TI Pharmacologic management of painful bladder syndrome/interstitial cystitis - A systematic review.
AU Dimitrakov, Jordan [Reprint Author]; Kroenke, Kurt; Steers, William D.; Berde, Charles; Zurakowski, David; Freeman, Michael R.; Jackson, Jeffrey L.
CS Harvard Univ, Sch Med, Childrens Hosp Boston, Harvard Urol Dis Res Ctr, Enders Res Bldg, Room 1061, 300 Longwood Ave, Boston, MA 02115 USA Jordan.Dimitrakov@childrens.harvard.edu
SO Archives of Internal Medicine, (OCT 8 2007) Vol. 167, No. 18, pp. 1922-1929. CODEN: AIMDAP. ISSN: 0003-9926.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 27 Dec 2007
Last Updated on STN: 27 Dec 2007
AB Background: More than 180 different types of therapy have been used in the treatment and management of painful bladder syndrome/interstitial cystitis (PBS/IC), yet evidence from clinical trials remains inconclusive. This study aimed to evaluate the efficacy of pharmacologic approaches to PBS/IC, to quantify the effect size from randomized controlled trials, and to begin to inform a clinical consensus of treatment efficacy for PBS/IC. Methods: We identified randomized controlled trials for the pharmacologic treatment of patients with PBS/IC diagnosed on the basis of National Institute of Diabetes and Digestive and Kidney Diseases or operational criteria. Study limitations include considerable patient heterogeneity as well as variability in the definition of symptoms and in outcome assessment. Results: We included a total of 1470 adult patients from 21 randomized controlled trials. Only trials for pentosan polysulfate sodium had sufficient numbers to allow a pooled analysis of effect. According to a random-effects model, the pooled estimate of the effect of pentosan polysulfate therapy suggested benefit, with a relative risk of 1.78 for patient-reported improvement in

symptoms (95% confidence interval, 1.34-2.35). This result was not heterogeneous (P=.47) and was without evidence of publication bias (P=.18). Current evidence also suggests the efficacy of dimethyl sulfoxide and amitryp- tiline therapy. Hydroxyzine, intravesical bacille Calmette-Guerin, and resinferatoxin therapy failed to demonstrate efficacy, but evidence was inconclusive owing to methodological limitations. Conclusions: Pentosan polysulfate may be modestly beneficial for symptoms of PBS/IC. There is insufficient evidence for other pharmacologic treatments. A consensus on standardized outcome measures is urgently needed.

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L1 31928 S CALCITONIN GENE RELATED PEPTIDE OR CGRP
L2 9041 S PITUITARY ADENYLATE CYCLASE ACTIVATING PEPTIDE OR
PACAP
L3 40576 S L1 OR L2
L4 43 S L3 AND INTERSTITIAL CYSTITIS
L5 27 DUP REM L4 (16 DUPLICATES REMOVED)

FILE 'STNGUIDE' ENTERED AT 17:23:30 ON 25 JAN 2010

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:35:03 ON 25 JAN 2010

L6 4413 S INTERSTITIAL CYSTITIS
L7 1735 S L6 AND DIAGNOS?
L8 316 S L7 AND REVIEW

FILE 'STNGUIDE' ENTERED AT 17:39:36 ON 25 JAN 2010

FILE 'BIOSIS, CAPLUS, EMBASE' ENTERED AT 17:41:39 ON 25 JAN 2010

=> s l3 and pelvic pain

L9 19 L3 AND PELVIC PAIN

=> dup rem l9

PROCESSING COMPLETED FOR L9

L10 12 DUP REM L9 (7 DUPLICATES REMOVED)

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AN 2009546163 EMBASE

TI Visceral hyperalgesia in chronic pelvic pain.

AU Aslam, N.; Harrison, G.; Khan, K.

CS Academic Department of Obstetrics and Gynaecology, Birmingham University

Hospital, Birmingham, United Kingdom.

AU Patwardhan, S. (correspondence)

CS Walsgrave University Hospital, Clifford Bridge Road, Coventry CV2 2DX,

United Kingdom. drsanjaypatwardhan@gmail.com

SO BJOG: An International Journal of Obstetrics and Gynaecology, (November

2009) Vol. 116, No. 12, pp. 1551-1555.

Refs: 25

ISSN: 1470-0328; E-ISSN: 1471-0528 CODEN: BIOGFQ

PB Blackwell Publishing Ltd, 9600 Garsington Road, Oxford, OX4 2XG,
United

Kingdom.

CY United Kingdom

DT Journal; Note

FS 005 General Pathology and Pathological Anatomy

008 Neurology and Neurosurgery

010 Obstetrics and Gynecology

028 Urology and Nephrology

037 Drug Literature Index

LA English

ED Entered STN: 18 Nov 2009

Last Updated on STN: 18 Nov 2009

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AN 2009489895 EMBASE

TI Endometriosis-associated nerve fibers and pain.

AU Medina, Melissa G.

CS University of Michigan, Ann Arbor, MI, United States.

AU Lebovic, Dan I.

CS Department of Obstetrics and Gynecology, Division of Reproductive
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SO Acta Obstetrica et Gynecologica Scandinavica, (2009) Vol. 88,

No. 9, pp.

968-975.

Refs: 29

ISSN: 0001-6349; E-ISSN: 1600-0412 CODEN: AOGSAE

PB Informa Healthcare, Telephone House, 69 - 77 Paul Street, EC2A

4LQ, United

Kingdom.

PUI 913657353

CY United Kingdom

DT Journal; General Review; (Review)

FS 005 General Pathology and Pathological Anatomy
010 Obstetrics and Gynecology
037 Drug Literature Index

LA English

SL English

ED Entered STN: 11 Nov 2009

Last Updated on STN: 11 Nov 2009

AB The assessment and diagnosis of endometriosis remain elusive targets.

Patient and medical-related factors add to delays in the detection and treatment. Recently, investigators have revealed specific nerve fibers

present in endometriotic tissue, with existing parallels between density

and pain severity. The aim of this review is to compile a comprehensive

review of existing literature on endometriosis-related nerve fiber

detection, and the effects of medical therapy on these neural fibers. We

performed a systematic literature-based review using Medline and PubMed of

nerve fibers detected in eutopic endometrium, endometriotic lesions, and

the peritoneum. Various arrangements of significant medical terms and

phrases consisting of endometriosis, pelvic pain,

nerve fiber detection/density in endometriosis, and diagnoses methodology,

including treatment and detection were applied in the search. Subsequent

references used were cross-matched with existing sources to compile all

additional similar reports. Similar nerve fibers were detected within

lesions, endometrium, and myometrium, though at varying degrees of

density. Hormonal therapy is widely used to treat endometriosis and was

shown to be related to a reduction in fiber density. A direct result of

specific nerve fiber detection within eutopic endometrial layers points to

the use of a minimally invasive endometrial biopsy technique in reducing

delay in diagnosis and subsequent possible preservation of fertility.

AN 2009:263278 BIOSIS
 DN PREV200900263278
 TI Rich innervation of deep infiltrating endometriosis.
 AU Wang, Guoyun; Tokushige, Natsuko [Reprint Author]; Markham, Robert;
 Fraser, Ian S.
 CS Univ Sydney, Queen Elizabeth II Res Inst Mothers and Infants,
 Dept Obstet
 and Gynecol, Sydney, NSW 2006, Australia
 ntokushige@med.usyd.edu.au
 SO Human Reproduction (Oxford), (APR 2009) Vol. 24, No. 4, pp.
 827-834.
 CODEN: HUREEE. ISSN: 0268-1161.
 DT Article
 LA English
 ED Entered STN: 16 Apr 2009
 Last Updated on STN: 16 Apr 2009
 AB Deep infiltrating endometriosis (DIE) is a specific type of
 endometriosis,
 which can be associated with more severe pelvic pain
 than other forms of endometriotic lesions. However, the
 mechanisms by
 which pain is generated are not well understood. DIE (n = 31) and
 peritoneal endometriotic (n = 40) lesions were sectioned and
 stained
 immunohistochemically with antibodies against protein gene
 product 9.5,
 neurofilament, nerve growth factor (NGF), NGF receptors tyrosine
 kinase
 receptor-A (Trk-A) and p75, substance P, calcitonin gene
 -related peptide, vesicular acetylcholine transporter,
 neuropeptide Y, vasoactive intestinal peptide and tyrosine
 hydroxylase to
 demonstrate myelinated, unmyelinated, sensory and autonomic nerve
 fibres. There were significantly more nerve fibres in DIE (67.6
 +/-
 65.1/mm(2)) than in peritoneal endometriotic lesions (16.3 +/-
 10.0/mm(2))
 (P < 0.01). DIE was innervated abundantly by sensory A delta,
 sensory C,
 cholinergic and adrenergic nerve fibres; NGF, Trk-A and p75 were
 strongly
 expressed in endometriotic glands and stroma of DIE. The rich
 innervation
 of DIE may help to explain why patients with this type of lesion
 have
 severe pelvic pain.

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 AN 2008361822 EMBASE

TI Evidence for the use of botulinum toxin in the chronic pain setting - A review of the literature.

AU Jeynes, Louise C.

CS The Boyle Department of Anesthesia, St. Bartholomew's Hospital, London, United Kingdom.

AU Gauci, Charles A., Dr. (correspondence)

CS Whipps Cross University Hospital, London, United Kingdom.
charles.gauci@btinternet.com

AU Gauci, Charles A., Dr. (correspondence)

CS Queen's Hospital, Essex, United Kingdom.
charles.gauci@btinternet.com

SO Pain Practice, (July/August 2008) Vol. 8, No. 4, pp. 269-276.
Refs: 100
ISSN: 1530-7085; E-ISSN: 1533-2500 CODEN: PPARCJ

PB Blackwell Publishing Inc., 350 Main Street, Malden, MA 02148, United States.

CY United States

DT Journal; (Short Survey)

FS 008 Neurology and Neurosurgery
019 Rehabilitation and Physical Medicine
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles

LA English

SL English

ED Entered STN: 7 Aug 2008
Last Updated on STN: 7 Aug 2008

AB A significant proportion of chronic pain is of musculoskeletal origin.
Botulinum toxin (BTX) has been successfully used in the treatment of spasmodic torticollis, limb dystonia, and spasticity. Investigators have, thus, become interested in its potential use in treating many chronic pain conditions. Practitioners have used BTX, outside the product license, in the treatment of refractory myofascial pain syndrome and neck and low back pain (LBP). This article reviews the current evidence relating to chronic pain practice. There is evidence supporting the use of both BTX type A and type B in the treatment of cervical dystonias. The weight of evidence is in favor of BTX type A as a treatment in: pelvic pain, plantar fasciitis, temporomandibular joint dysfunction associated facial

pain, chronic LBP, carpal tunnel syndrome, joint pain, and in complex regional pain syndrome and selected neuropathic pain syndromes. The weight of evidence is also in favor of BTX type A and type B in piriformis syndrome. There is conflicting evidence relating to the use of BTX in the treatment of whiplash, myofascial pain, and myogenous jaw pain. It does appear that BTX is useful in selected patients, and its duration of action may exceed that of conventional treatments. This seems a promising treatment that must be further evaluated. .COPYRGHT. 2008 World Institute of Pain.

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AN 2007507114 EMBASE
TI Neuroendocrine-immune disequilibrium and endometriosis: An interdisciplinary approach.
AU Tariverdian, Nadja; Blois, Sandra M.; Arck, Petra C.
(correspondence)
CS Center of Internal Medicine and Dermatology, Division of PsychoNeuroImmunology, University Medicine Berlin, Berlin, Germany.
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AU Theoharides, Theoharis C.
CS Department of Pharmacology and Experimental Therapeutics, Tufts University School of Medicine, Boston, MA, United States.
AU Siedentopf, Friederike
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AU Gutierrez, Gabriela
CS Institute of Humoral Immunity Studies-IDEHU (CONICET-UBA), School of Pharmacy and Biochemistry, University of Buenos Aires, Buenos Aires, Argentina.
AU Jeschke, Udo
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 AU Arck, Petra C. (correspondence)
 CS Biomedizinisches Forschungszentrum, Charite, Campus Virchow,
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 petra.arck@charite.de
 SO Seminars in Immunopathology, (Jun 2007) Vol. 29, No. 2, pp.
 193-210.
 Refs: 196
 ISSN: 1863-2297; E-ISSN: 1863-2300
 CY Germany
 DT Journal; General Review; (Review)
 FS 010 Obstetrics and Gynecology
 026 Immunology, Serology and Transplantation
 005 General Pathology and Pathological Anatomy
 LA English
 SL English
 ED Entered STN: 2 Nov 2007
 Last Updated on STN: 2 Nov 2007
 AB Endometriosis, a chronic disease characterized by endometrial
 tissue
 located outside the uterine cavity, affects one fourth of young
 women and
 is associated with chronic pelvic pain and
 infertility. However, an in-depth understanding of the
 pathophysiology
 and effective treatment strategies of endometriosis is still
 largely
 elusive. Inadequate immune and neuroendocrine responses are
 significantly
 involved in the pathophysiology of endometriosis, and key
 findings are
 summarized in the present review. We discuss here the role of
 different
 immune mechanisms particularly adhesion molecules, protein-glycan
 interactions, and pro-angiogenic mediators in the development and
 progression of the disease. Finally, we introduce the concept of
 endometrial dissemination as result of a neuroendocrine-immune
 disequilibrium in response to high levels of perceived stress
 caused by
 cardinal clinical symptoms of endometriosis. .COPYRGT. 2007
 Springer-Verlag.

L10 ANSWER 6 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson
 Corporation on STN

DUPLICATE 2

AN 2007:39911 BIOSIS
 DN PREV200700041566
 TI Nerve fibres in peritoneal endometriosis.
 AU Tokushige, Natsuko [Reprint Author]; Markham, Robert; Russell,
 Peter;

Fraser, Ian S.

CS Univ Sydney, Dept Obstet and Gynaecol, Queen Elizabeth Res Inst Mothers

and Infants 2, Sydney, NSW 2006, Australia

ntokushige@med.usyd.edu.au

SO Human Reproduction (Oxford), (NOV 2006) Vol. 21, No. 11, pp. 3001-3007.

CODEN: HUREEE. ISSN: 0268-1161.

DT Article

LA English

ED Entered STN: 3 Jan 2007

Last Updated on STN: 3 Jan 2007

AB BACKGROUND: Endometriosis is a gynaecological disease that can be associated with severe pelvic pain; however, the mechanisms by which pain is generated remain unknown. METHODS:

Peritoneal

endometriotic lesions and normal peritoneum were prepared from women with

and without endometriosis (n = 40 and 36, respectively).

Specimens were

also prepared from endosalpingiosis lesions (n = 9). These sections were

stained immunohistochemically with antibodies against protein gene product

9.5, neurofilament (NF), nerve growth factor (NGF), NGF receptor p75

(NGFRp75), substance P (SP), calcitonin gene-

related peptide (CGRP), acetylcholine (ACh)

and tyrosine hydroxylase (TH) to demonstrate myelinated, unmyelinated,

sensory, cholinergic and adrenergic nerve fibres. RESULTS:

There were

significantly more nerve fibres identified in peritoneal

endometriotic

lesions than in normal peritoneum (P < 0.001) or

endosalpingiosis lesions

(P < 0.001). These nerve fibres were SP, CGRP, ACh or TH

immunoreactive. Many of these markers were co-localized. There was an

intense NGF immunoreactivity near endometriotic glands, and NGFRp75

immunoreactive nerve fibres were present near endometriotic glands and

blood vessels in the peritoneal endometriotic lesions.

CONCLUSIONS:

Peritoneal endometriotic lesions were innervated by sensory A

delta,

sensory C, cholinergic and adrenergic nerve fibres. These nerve fibres

may play an important role in the mechanisms of pain generation in this

condition.

L10 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2010 ACS on STN
 AN 2005:405354 CAPLUS
 DN 142:461621
 TI Detection of neuropeptides associated with pelvic pain
 disorders and uses for diagnosis and treatment
 IN Wood, Ronald W.; Reeder, Jay; Schwarz, Edward M.; Messing,
 Edward M.;
 Schoen, Susan R.; Vizzard, Margaret A.; Dickerson, Ian
 PA University of Rochester, USA
 SO PCT Int. Appl., 43 pp.
 CODEN: PIXXD2

DT Patent
 LA English

FAN.CNT 1

DATE	PATENT NO.	KIND	DATE	APPLICATION NO.
PI	WO 2005041757	A2	20050512	WO 2004-US36015
20041029	WO 2005041757	A3	20060601	
CA, CH,	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ,			
GB, GD,	CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI,			
KZ, LC,	GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR,			
NA, NI,	LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ,			
SL, SY,	NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK,			
ZM, ZW	TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA,			
ZW, AM,	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM,			
DE, DK,	AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ,			
RO, SE,	EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT,			
MR, NE,	SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,			
	SN, TD, TG			
	US 20080070239	A1	20080320	US 2007-577395

20070427

PRAI US 2003-515408P P 20031029
 WO 2004-US36015 W 20041029

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
 AB The present invention relates generally to the diagnosis and
 treatment of

pelvic pain disorders, including bladder disorders that
 are characterized by increased expression of the neuropeptides

CGRP and/or PACAP. One aspect of the present invention is directed to a method of diagnosing pelvic pain disorders. This method involves measuring a level of one or both of the neuropeptides calcitonin gene-related peptide (CGRP) or pituitary adenylate cyclase activating peptide (PACAP) in a patient sample and then determining whether the CGRP or PACAP level in the patient sample is elevated in relation to a level of CGRP or PACAP in a normal asymptomatic population. A second aspect of the present invention is directed to a method of determining predisposition of an individual to conditions associated with or development of pelvic pain syndromes. A third aspect of the present invention is directed to a method of treating a pelvic pain disorder in a patient. This method involves providing a CGRP or PACAP antagonist and administering the CGRP or PACAP antagonist to the patient in an amount effective to treat the pelvic pain disorder. A fourth aspect of the present invention is directed to a method of characterizing response to treatment for a pelvic pain disorder. A fifth aspect of the present invention relates to a transgenic nonhuman mammal that includes a first DNA construct that is expressed in bladder sensory neurons, the first DNA construct having a promoter operatively coupled to a DNA mol. encoding a neuropeptide (either PACAP or CGRP). The transgenic nonhuman mammals are characterized by overexpression (i.e., relative to nontransgenic mammals) of the neuropeptide. These transgenic animals are useful for the study of pelvic pain disorders and assessing the efficacy of potential therapeutic agents in the treatment thereof.

OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L10 ANSWER 8 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 3

AN 2006:27247 BIOSIS

DN PREV200600024144

TI Possible mechanism of referred pain in the perineum and pelvis associated

with the prostate in rats.

AU Chen, Yong; Song, Bo [Reprint Author]; Jin, Xi-Yu; Xiong, En-Qing; Zhang, Jian-Hua

CS Third Mil Med Univ, SW Hosp, Dept Urol, Chongqing 40008, Peoples R China

SO Journal of Urology, (DEC 2005) Vol. 174, No. 6, pp. 2405-2408.
CODEN: JOURAA. ISSN: 0022-5347.
DT Article
LA English
ED Entered STN: 21 Dec 2005
Last Updated on STN: 21 Dec 2005
AB Purpose: Since persistent pain in the perineum and pelvic floor associated with chronic prostatitis/chronic pelvic pain syndrome has been hypothesized to be referred pain, it might also be explained by neural mechanisms. Materials and Methods: Dual retrograde fluorescent labeling and immunohistochemistry were identified as methods with which to investigate the neurogenic aspect of this status. The dual distribution of dorsal root ganglia (DRG) cells was determined after double retrograde fluorescent staining of the prostate and pelvic floor, and the prostate and perineum somatic nerves. Calcitonin gene-related peptide (CGRP) and substance P (SP) in dual labeled cells were determined by immunohistochemistry, giving possible insight into the cause of pelvic pain. Results: Fluorescent double labeled cells were found in the lumbar and sacral DRG, while double labeled cells were distributed predominantly in L6 to S1 and L1 to L2 segment DRG in groups 1 and 2, respectively. On immunohistochemistry some of them were confirmed to contain CGRP and SP. Thus, there are crossover pathways between the prostate and pelvic floor. Conclusions: The findings that we present confirm that the peripheral process of DRG cells dichotomizes to the prostate, sphincter and somatic parties simultaneously. Some of these cells contain CGRP and SP, which indicate that referred pain in the perineum and pelvic floor may be caused by an axon reflex in the peripheral process of DRG neurons.

L10 ANSWER 9 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN

DUPLICATE 4

AN 2004:398362 BIOSIS

DN PREV200400399374

TI Innervation of ectopic endometrium in a rat model of endometriosis.

AU Berkley, Karen J. [Reprint Author]; Dmitrieva, Natalia; Curtis, Kathleen
S.; Papka, Raymond E.
CS Program Neurosci, Florida State Univ, Tallahassee, FL, 32306, USA
kberkley@psy.fsu.edu
SO Proceedings of the National Academy of Sciences of the United States of America, (July 27 2004) Vol. 101, No. 30, pp. 11094-11098.
print.
ISSN: 0027-8424 (ISSN print).
DT Article
LA English
ED Entered STN: 13 Oct 2004
Last Updated on STN: 13 Oct 2004
AB Endometriosis (ENDO) is a disorder in which vascularized growths of endometrial tissue occur outside the uterus. Its symptoms include reduced fertility and severe pelvic pain. Mechanisms that maintain the ectopic growths and evoke symptoms are poorly understood. One factor not yet considered is that the ectopic growths develop their own innervation. Here, we tested the hypothesis that the growths develop both an autonomic and a sensory innervation. We used a rat model of surgically induced ENDO whose growths mimic those in women. Furthermore, similar to women with ENDO, such rats exhibit reduced fertility and increased pelvic nociception. The ENDO was induced by autotransplanting, on mesenteric cascade arteries, small pieces of uterus that formed vascularized cysts. The cysts and healthy uterus were harvested from proestrous rats and immunostained using the pan-neuronal marker PGP9.5 and specific markers for calcitonin gene-related peptide (CGRP) (sensory C and AS fibers), substance P (SIP) (sensory C and AS fibers) and vesicular monoamine transporter (sympathetic fibers). Cysts (like the uterus) were robustly innervated, with many PGP9.5-stained neurites accompanying blood vessels and extending into nearby luminal epithelial layers. CGRP-, SP-, and vesicular monoamine transporter-immunostained neurites also were observed, with CGRP and SP neurites extending the furthest into the cyst lining. These results demonstrate that ectopic endometrial growths

develop an autonomic and sensory innervation. This innervation could contribute not only to symptoms associated with ENDO but also to maintenance of the ectopic growths.

L10 ANSWER 10 OF 12 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights

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AN 2004355964 EMBASE

TI Mechanisms in prostatitis/chronic pelvic pain syndrome.

AU Pontari, Michel A. (correspondence); Ruggieri, Michael R.

CS Department of Urology, Temple University School of Medicine, Philadelphia,

PA, United States. Pontarm@tuhs.temple.edu

AU Pontari, Michel A. (correspondence)

CS Department of Urology, Temple University, Parkinson Pavilion, 3401 North

Broad St., Philadelphia, PA 19140, United States.

Pontarm@tuhs.temple.edu

SO Journal of Urology, (Sep 2004) Vol. 172, No. 3, pp. 839-845. Refs: 75

ISSN: 0022-5347 CODEN: JOURAA

CY United States

DT Journal; General Review; (Review)

FS 026 Immunology, Serology and Transplantation

028 Urology and Nephrology

003 Endocrinology

008 Neurology and Neurosurgery

LA English

SL English

ED Entered STN: 16 Sep 2004

Last Updated on STN: 16 Sep 2004

AB Purpose: We reviewed the current literature on mechanisms involved in the

pathogenesis of prostatitis/chronic pelvic pain

syndrome (CPPS). Materials and Methods: A literature review for the years

1966 to 2003 was performed using the MEDLINE database of the United States

National Library of Medicine. Results: National Institutes of Health

categories I and II prostatitis result from identifiable prostatic

infections, whereas patients with category IV are asymptomatic.

The

majority of symptomatic cases are category III or chronic

prostatitis

(CP)/CPPS. The etiology of CP/CPPS is unknown. The traditional marker of

inflammation, namely white blood cells in prostatic fluids, does

not

correlate with the predominant symptom of pelvic pain. An imbalance toward increased proinflammatory and decreased anti-inflammatory cytokines has been implicated and a few studies have shown some correlation of this with pelvic pain. The imbalance in some men may result from polymorphisms at the cytokine loci. An autoimmune process may be involved and experimental evidence indicates that this can be under hormonal influence. Recent findings include possible defects in the androgen receptor. The prostate may not even be the source of the symptoms. Pelvic pain also correlates with the neurotrophin nerve growth factor implicated in neurogenic inflammation and central sensitization. Finally, psychological stress may produce measurable biochemical changes and influence the other processes. The role of normal prostatic bacterial flora in inciting the inflammatory response has also been reconsidered. Conclusions: The symptoms of CP/CPPS appear to result from an interplay between psychological factors and dysfunction in the immune, neurological and endocrine systems.

L10 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2010 ACS on STN
 AN 2002:256747 CAPLUS
 DN 136:257266
 TI Methods of diagnosing and treating small intestinal bacterial overgrowth and related conditions
 IN Lin, Henry C.; Pimentel, Mark
 PA USA
 SO U.S. Pat. Appl. Publ., 53 pp., Cont.-in-part of U. S. Ser. No. 374,142.
 CODEN: USXXCO
 DT Patent
 LA English
 FAN.CNT 6

DATE	PATENT NO.	KIND	DATE	APPLICATION NO.
-----	-----	----	-----	-----
PI 20010417	US 20020039599	A1	20020404	US 2001-837797
	US 7048906	B2	20060523	
	CA 2220451	A1	19961121	CA 1996-2220451
19960516				

US 5977175	A	19991102	US 1997-832307
19970403			
US 6562629	B1	20030513	US 1999-374143
19990811			
US 6861053	B1	20050301	US 1999-374142
19990811			
US 20020094346	A1	20020718	US 1999-420046
19991018			
US 6558708	B1	20030506	US 2000-546119
20000410			
WO 2001011334	A2	20010215	WO 2000-US22168
20000811			
WO 2001011334	A3	20010712	
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA,			
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CH, CY,			
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	CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG		
EP 1811303	A2	20070725	EP 2007-75358
20000811			
EP 1811303	A3	20070815	
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI,			
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	NL, PT, SE		
CA 2444548	A1	20021024	CA 2002-2444548
20020416			
WO 2002083926	A2	20021024	WO 2002-US12034
20020416			
WO 2002083926	A3	20030515	
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA,			
CH, CN,			
	CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD,		
GE, GH,			
	GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,		
LK, LR,			
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TT, TZ,			
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SE, TR,				
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TD, TG				
AU 2002256254	A1	20021028	AU 2002-256254	
20020416				
AU 2002256254	B2	20070118		
EP 1385476	A2	20040204	EP 2002-725704	
20020416				
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE,				
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JP 2005509588	T	20050414	JP 2002-582263	
20020416				
US 20040180834	A1	20040916	US 2004-810020	
20040326				
US 7081239	B2	20060725		
US 20050014693	A1	20050120	US 2004-853824	
20040526				
US 7244412	B2	20070717		
US 20050008652	A1	20050113	US 2004-915193	
20040810				
US 7056686	B2	20060606		
US 20060029550	A1	20060209	US 2005-234516	
20050923				
US 7452857	B2	20081118		
US 20060147496	A1	20060706	US 2006-348995	
20060207				
US 20060193871	A1	20060831	US 2006-411733	
20060425				
US 20060246085	A1	20061102	US 2006-457445	
20060713				
US 7608245	B2	20091027		
AU 2007200008	A1	20070125	AU 2007-200008	
20070102				
AU 2007200008	B2	20080911		
US 20070142291	A1	20070621	US 2007-673488	
20070209				
US 7615207	B2	20091110		
AU 2007201246	A1	20070419	AU 2007-201246	
20070322				
AU 2007201246	B2	20081204		
US 20080014184	A1	20080117	US 2007-838631	
20070814				
US 7585838	B2	20090908		
US 20080014185	A1	20080117	US 2007-838672	
20070814				
US 7605240	B2	20091020		
US 20090012113	A1	20090108	US 2008-234502	
20080919				
JP 2009102401	A	20090514	JP 2009-16943	
20090128				

US 20090325994	A1	20091231	US 2009-550303
20090828			
PRAI US 1995-442843	B1	19950517	
US 1997-832307	A1	19970403	
US 1999-359583	B2	19990722	
US 1999-374142	A2	19990811	
US 1999-374143	A2	19990811	
US 1999-420046	A2	19991018	
US 2000-546119	A2	20000410	
EP 2000-952739	A3	20000811	
WO 2000-US22030	A	20000811	
WO 2000-US22168	A	20000811	
AU 2001-251396	A3	20010407	
WO 2001-US11238	A	20010407	
US 2001-837797	A	20010417	
US 2002-107240	A3	20020326	
AU 2002-256254	A3	20020416	
JP 2002-582263	A3	20020416	
WO 2002-US12034	W	20020416	
US 2004-810020	A1	20040326	
US 2004-853824	A3	20040526	
US 2004-915193	A1	20040810	
US 2005-234516	A3	20050923	
US 2006-457445	A1	20060713	
US 2007-838672	A1	20070814	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Disclosed is a method of treating small intestinal bacterial overgrowth

(SIBO) or a SIBO-caused condition in a human subject.

SIBO-caused

conditions include irritable bowel syndrome, fibromyalgia, chronic

pelvic pain syndrome, chronic fatigue syndrome, depression, impaired mentation, impaired memory, halitosis, tinnitus, sugar craving, autism, attention deficit/hyperactivity disorder, drug

sensitivity, an autoimmune disease, and Crohn's disease.

Examples are

provided showing effects of antibiotics on SIBO, demonstrating the roles

of peptide YY and the serotonergic/adrenergic/opioid pathways in SIBO,

and the effects of ondansetron, propranolol, norepinephrine and naloxone

on intestinal transit. The invention thus relates to slowing upper

gastrointestinal transit, thereby enhancing the digestion and/or absorption of predigested nutrients. Gastrointestinal transit-slowing

comps. comprise active agents such as lipids, serotonin, serotonin

agonists, serotonin re-uptake inhibitors, peptide YY, calcitonin gene-related peptide, adrenergic agonists and opioid agonists. Also disclosed are a method of screening for the abnormally likely presence of SIBO in a human subject and a method of detecting SIBO in a human subject. A method of determining the relative severity of SIBO or a SIBO-caused condition in a human subject, in whom small intestinal bacterial overgrowth has been detected, is also disclosed.

OSC.G 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

RE.CNT 251 THERE ARE 251 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 12 OF 12 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 5

AN 2001:24473 BIOSIS

DN PREV200100024473

TI Growth of nerve fibres into murine peritoneal adhesions.

AU Sulaiman, Hassan; Gabella, Giorgio; Davis, Christine; Mutsaers, Steven E.; Boulos, Paul; Laurent, Geoffrey J.; Herrick, Sarah E. [Reprint author]

CS Department of Medicine, Rayne Institute, University College London, 5 University Street, London, WC1E 6JJ, UK
s.herrick@ucl.ac.uk

SO Journal of Pathology, (November, 2000) Vol. 192, No. 3, pp. 396-403.
print.
CODEN: JPTLAS. ISSN: 0022-3417.

DT Article

LA English

ED Entered STN: 3 Jan 2001
Last Updated on STN: 12 Feb 2002

AB Adhesions in the peritoneal cavity have been implicated in the cause of intestinal obstruction and infertility, but their role in the aetiology of chronic pelvic pain is unclear. Nerves have been demonstrated in human pelvic adhesions, but the presence of pain-conducting fibres has not been established. The purpose of this study was to use an animal model to examine the growth of nerves during adhesion formation at various times following injury and to characterize the types of fibres present. Adhesions were generated in mice by injuring

the surface of the caecum and adjacent abdominal wall, with apposition.

At 1-8 weeks post-surgery, adhesions were processed and nerve fibres characterized histologically, immunohistochemically, and ultrastructurally. Peritoneal adhesions had consistently formed by 1 week after surgery and from 2 weeks onwards, all adhesions contained some nerve fibres which were synaptophysin, calcitonin gene-related peptide, and substance P-immunoreactive, and were seen to originate from the caecum. By 4 weeks post-surgery, nerve fibres were found to originate from both the caecum and the abdominal wall, and as demonstrated by acetylcholinesterase histochemistry, many traversed the entire adhesion. Ultrastructural analysis showed both myelinated and non-myelinated nerve fibres within the adhesion. This study provides the first direct evidence for the growth of sensory nerve fibres within abdominal visceral adhesions in a murine model and suggests that there may be nerve fibres involved in the conduction of pain stimuli.

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